I hereby certify that, on the date shown below, this correspondence is being:
☐ deposited with the United States Postal Service in an envelope addressed to: ASSISTANT COMMISSIONER FOR PATENTS, WASHINGTON, D.C. 20231,
☐ under 37CFR § 1.8(a), with sufficient postage as first class mail, or
☐ under 37CFR § 1.10, as "Express Mail Post Office to Addressee" Mailing Label No. _____,
☒ transmitted by facsimile to the Patent and Trademark Office, Fax Number 703-872-9306, Attention: Examiner J. Andres, Art Unit 1646

Date: December 11, 2002 By: Lois Miller

PATENT

Attorney Docket No. DX01073K

CN 028008

#12
B.Q.J
12/30/02

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re application of:

Debets, et al.

Serial No.: 09/775,046

Filed: February 1, 2001

For: MAMMALIAN CYTOKINES; RECEPTORS; RELATED REAGENTS AND METHODS

Examiner: J. Andres

Art Unit: 1646

RESPONSE TO RESTRICTION REQUIREMENT

Palo Alto, California 94304

December 11, 2002

Assistant Commissioner for Patents
Washington, D.C. 20231

Honorable Sir:

This is a response to the Restriction Requirement dated November 18, 2002 (Paper 11).

The Examiner restricted the application into ten separate inventions:

I. Claims 1-3, drawn to methods of producing a ligand-receptor complex, classified in class 435, subclass 7.1.

II. Claims 4-11, drawn to methods of modulation of the IL-1R6 receptor, classified in class 514, subclass 2.

III. Class 12-15, drawn to methods of identifying and purifying cells, and cells obtained by these methods, classifi d in class 435, subclasses 7.1 and 325.

IV. Claims 16 and 17, drawn to screening methods, classified in class 435, subclass 7.1.

V. Claim 18, drawn to polynucleotides encoding SEQ ID NO:2, classified in class 435, subclass 69.1.

VI. Claim 18, drawn to polynucleotides encoding SEQ ID NO:4, classified in class 435, subclass 69.1.

VII. Claim 19, drawn to the polypeptide of SEQ ID NO:2, classified in class 530, subclass 351.

VIII. Claim 19, drawn to the polypeptide of SEQ ID NO:4, classified in class 530, subclass 351.

IX. Claim 20, drawn to antibodies against the polypeptide of SEQ ID NO:2, classified in class 530, subclasses 388.1 and 389.1.

X. Claim 20, drawn to antibodies against the polypeptide of SEQ ID NO:4, classified in class 530, subclasses 388.1 and 389.1.

Where Groups I, III, or IV are elected, the Examiner further imposed a species selection from the species IL-1delta and IL-1epsilon. Where Group II is elected, the Examiner further imposed a species selection from (1) IL-1delta and IL-1epsilon; (2) Antagonist and agonist; (3) Proliferation, tissue remodeling, and inflammation; and (4) One of the following co-administered agents: none, chemokine receptor antagonist, chemokine receptor agonist, growth factor or cytokine, chemokine, and immun- adjuvant.

Applicants elect Group II, drawn to methods of modulation of the IL-1R6 receptor. Group II comprises Claims 4-11, as filed. Applicants provisionally select, with traverse, the following species: IL-1epsilon; antagonist; inflammation; and co-administered chemokine receptor antagonist.

Applicants traverse the species selection on the grounds that no serious burden would exist to examine proliferation and tissue remodeling together with inflammation. Applicants submit that proliferation and tissue remodeling are subsets of inflammation. According to the current edition of Rich, et al., inflammation may include tissue remodeling as well as cell proliferation (Rich, et al. (eds.) (2001) Clinical Immunology, Volume One, Mosby, New York, pp. 3.4 & 16.1) (enclosed). Proliferation is a part of the inflammatory process, because "[i]n order for T cells to effectively mediate inflammation they must . . . expand in number . . ." (page 16.1, column 2, of Rich, et al., supra). Tissue remodeling is part of the inflammatory process, because "marked histological manifestations become apparent in affected nonlymphoid tissues." (page 3.4, column 2, of Rich, et al., supra). Rich, et al., is cited in the Specification (page 65, lines 17-18). Rejoinder of the species inflammation, proliferation, and tissue remodeling is respectfully requested.

Applicants also traverse the species selection requirement on the grounds that no serious burden would exist to examine the species of "no co-administered agent" with the elected species of co-administered agent of "chemokine receptor antagonist." Rejoinder of these species is respectfully requested.

A list of all unamended claims that read upon the elected species appears in the Appendix.

Applicants will address the issue of inventorship for the elected claims and amend inventorship appropriately if the Restriction Requirement should be made final.

Applicants reserve the right to file subsequent applications claiming the non-elected subject matter and do not waive any of their rights or abandon any non-elected subject matter. Since Applicants have fully and completely responded to the Restriction Requirement and have made the required election, this application is now in order for early action.

Applicants believe that no fees are due with this communication. Should this not be the case, the Commissioner is hereby authorized to debit any charges or refund any overpayments to DNAX Deposit Account No. 04-1239. If the Examiner believes that a telephonic conference would aid the prosecution of this case in any way, please call the undersigned.

Respectfully submitted,

Date: December 11, 2002

By: _____
Sheela Mohan-Peterson
Attorney for Applicants
Reg. No. 41,201

DNAX Research, Inc.
901 California Avenue
Palo Alto, California 94304-1104
Tel No.: (650) 496-6400
Fax: (650) 496-1200

Enclosed:

Rich, et al. (eds.) (2001) Clinical Immunology, Volume One, Mosby, New York, pp. 3.4 & 16.1

APPENDIX

The following is a list of all unamended claims that read upon the elected species:

4. A method of modulating physiology or development of an IL-1R6 receptor expressing cell comprising contacting said cell to an exogenous agonist or antagonist of a mammalian IL-1δ or IL-1ε.

5. The method of Claim 4, wherein:
   A) said antagonist is:
      1) an antibody which:
         a) neutralizes said mammalian IL-1δ; or
         b) neutralizes said mammalian IL-1ε; or
      2) a mutein of said IL-1δ or IL-1ε;
   B) said physiology is selected from:
      1) proliferation;
      2) tissue remodeling; or
      3) production of inflammatory mediators, including cytokines, chemokines, or adhesion molecules; or
   C) said modulating is specific for epithelial cells and not endothelial cells.

6. The method of Claim 4, wherein:
   a) said antagonist is an antibody and said physiology is an inflammatory response; or
   b) said modulating is specific for Th2 cells and not Th1 cells.

7. The method of Claim 4, wherein said modulating is blocking, and said physiology is an inflammatory response.

8. A method of modulating a signal to a cell mediated by IL-1δ or IL-1ε comprising contacting said cell to an administered agonist or antagonist of IL-1R6.

9. The method of Claim 8, wherein said modulating is inhibiting, and said signal is a pro-inflammatory signal.

10. The method of Claim 9, wherein:

a) said antagonist is a neutralizing antibody to IL-1R6;

b) said agonist or antagonist is administered in combination with an antagonist or agonist of CXCR1, CXCR2, or CCR6; or

c) said agonist or antagonist is administered in combination with a growth factor, cytokine, chemokine, or immune adjuvant.

11. The method of Claim 9, wherein said contacting is with another anti-inflammatory agent.